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REVIEW

Aneurysms of the visceral and renal arteries

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Visceral aneurysms represent a rare clinical entity; however, 10–20% will rupture and this is accompanied by a significant mortality rate of 20–70%, depending on the location of the aneurysm. The incidence, pathogenesis and clinical aspects of splanchnic and renal artery aneurysms are reviewed from the available literature and the problems of diagnosis and treatment are discussed. Their incidence is increasing and controversy still exists regarding their treatment. The decision for intervention has to take into account the size and the natural history of the lesion, the risk of rupture, which is high during pregnancy, and the relative risk of surgical or radiological intervention. For most asymptomatic aneurysms, expectant treatment is acceptable. For large, symptomatic or aneurysms with a high risk of rupture, surgery is advisable. An alternative treatment is the use of endovascular techniques, ie embolisation, or graft stent insertion.

Aneurysms of the visceral arteries are a rarity, which most doctors will never encounter. Their prevalence is estimated to be around 1% of the population *(1–6)*. However, with the advent of computed tomography and arteriography, the incidental finding of visceral aneurysms has increased. More than 3000 cases were reported in the literature up to 1992 *(3)* with a further 213 aneurysms since then (103 splenic, 70 renal, 15 of the coeliac axis and its branches, 13 of the superior mesenteric and 3 of the inferior mesenteric artery), an average of 70 splanchnic aneurysms per year. Most visceral aneurysms are asymptomatic; however, they may present as life-threatening intra-abdominal apoplexy. Despite their rarity, they represent a serious vascular condition, as about 25% will rupture *(3,4)*; if they do, the mortality varies from 25% to 70%, according to their location, corresponding to a total mortality of 8–10% for the total number of splanchnic aneurysms *(1–7)*.

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Incidence

The incidence in post-mortem reports varies, from 0.098% *(4)* to 10.4% *(8)*, but on average is 1% *(1–3)*. Splenic artery aneurysms are second in frequency to aneurysms of the aortoiliac segment in the abdomen. About 60% of the splanchnic aneurysms involve the splenic artery *(1–6,8,9)*, 20% the hepatic *(10–12)*, 5% the superior mesenteric artery *(1–4,13–19)*, 4% the smaller branches of the coeliac artery (gastric, pancreatoduodenal, gastroepiploic *(3,4,6,20–25)*, and another 4% the coeliac artery *(1,6,12)*. Jejunal, ileal and colic arteries represent 3% of the total number *(1–6,26–31)*.

Renal artery aneurysms are usually described separately, as they show a slightly different natural history, with a significantly lower risk of rupture and considerably lower mortality rates. They are strongly associated with hypertension. The incidence of true renal aneurysms, excluding microaneurysms and dissections, is approximately 0.1% *(32–36)*. In a review of 10 000 aortograms, the prevalence was 0.3% *(34)*, while in 8525 renal angiograms only 83 (0.1%) aneurysms were reported *(36)*.

Splenic artery aneurysms are four times more common in women *(4–6,8,9)*, while hepatic artery aneurysms are more common in men (ratio 2:1) *(10,11,37–39)*; gastric, gastroepiploic and pancreatoduodenal artery aneurysms

are 3–4 times more common in men *(1,3,6,21)*, while renal artery aneurysms have an equal incidence in men and women *(3,32–36)*.

Aetiology

The cause of visceral aneurysms is controversial. Although atherosclerosis is frequently cited as the most common cause, Stanley and Fry *(5)* proposed that the typical calcific atherosclerotic changes in many of these aneurysms is more likely to be a secondary event rather than the primary aetiological process.

The majority probably result from an abnormality in the medial arterial wall, which may be congenital in origin, and this is further stressed by various acquired factors. Another cause is the presence of inflammation either in the arterial walls, eg polyarteritis nodosa, other vasculitis *(40–43)* mycotic aneurysms secondary to infective endocarditis *(13,19)*, or the adjacent tissues, eg pancreatitis *(22,24,25,44,45)*, which erode into the artery, forming a false aneurysm. Recently, there is an increased recognition of aneurysms of the splenic and coeliac artery branches after liver transplantation *(46,47)*. Trauma, although reported, is probably a rare cause *(13,48)*.

Splenic artery aneurysms

The most common factors are increased splenic blood flow and the increased activity of oestrogens which seem to have an adverse effect on the elastic fibres of the vascular wall *(23,49)*. Of women with splenic aneurysms, 40% are multiparous *(1–5,50–52)* and 10% of patients with portal hypertension and cirrhosis harbour splenic artery aneurysms *(3,5,9,49,53)*. Fibrodysplasia is also a common factor, as 2% of patients with renal fibrodysplasia present with splenic aneurysms *(3)*. Pancreatitis, vasculitis and trauma represent less common causes *(1–6)*.

Hepatic artery aneurysms

There is no uniform cause and their aetiology and pathogenesis is poorly defined. Polyarteritis nodosa is associated *(41,42)*, but neither vasculitic nor iatrogenic false aneurysms after biliary tract procedures are common *(3,5,12)*. Atherosclerosis is more likely a secondary event, while the incidence of traumatic and mycotic aneurysms seems to be increasing *(10–12,54)*.

Superior mesenteric artery

Most of them are mycotic, secondary to bacterial endocarditis or drug abuse *(7,13,14)*. Medial degeneration *(1,17)*, trauma *(1–3)*, and Bechet's disease *(15,16)* have also been reported.

Coeliac artery, gastric and gastroepiploic artery aneurysms

These are usually attributed to infection or medial wall degeneration *(7)*. Vasculitis *(7,42)* and infective endocarditis *(18)* have also been reported.

Intestinal branch arteries (jejunal, ileal, colic)

They are secondary either to septic emboli (infective endocarditis) or polyarteritis nodosa and other connective tissue disorders *(18,53,55)*.

Inferior mesenteric artery aneurysms

These are so rare that no speculation can be made about their aetiology *(56,57)*.

Pancreatoduodenal and gastroduodenal arteries

The most common cause (60%) is pancreatitis with erosion of a pseudocyst into the vessel *(6,21–24,45)*

Renal artery aneurysms

They are usually attributed to medial degeneration and fibrodysplasia *(32,58,59)* as the most common histopathological finding is fragmentation of the internal elastic lamina. Hypertension may also contribute to some degree *(3,36)*. Atherosclerotic lesions are usually present; however, the lack of similar findings in the adjacent vessels suggests that atherosclerosis is not a causative process *(32)*. Neurofibromatosis has also been associated with multiple aneurysms of the branches of the renal arteries *(60–62)*. Aneurysms secondary to arteritis are extremely rare *(43)*.

Natural history and diagnosis

The majority of visceral aneurysms are asymptomatic *(1–6)* with the exception of gastric *(7)* pancreatoduodenal *(22,26)* and superior mesenteric artery aneurysms *(13–18,26,63,64)*. Renal artery aneurysms are associated with hypertension *(32,34,58,59,65,66)*, but it is not certain if they are a cause or an effect of hypertension.

About 60% of visceral aneurysms are discovered incidentally, through plain radiographs, angiography or computed tomography undertaken for some other reason *(34,67)*. Of the remainder, 20% become symptomatic and another 20% present as acute rupture *(1–6)*, particularly during pregnancy *(3,26,68,69)*.

Splenic artery aneurysms (Figs 1–3)

Most are saccular, less than 2 cm in diameter (80%) *(1–7, 70)* and occur where the artery branches *(1,3,5,6)*, while those associated with pancreatitis involve the main trunk of the vessel *(44,45,71)*. The majority are asymptomatic and left quadrant pain occurs in only a minority. Rupture occurs in about 2% of asymptomatic patients *(3,5,9,26)*. Bleeding is first contained in the lesser sac and is accompanied by epigastric pain; as blood passes through the foramen of Winslow and enters the right paracolic gutter, the pain moves to the right upper and lower quadrants; eventually, vascular collapse follows intraperitoneal haemorrhage. This is called the double rupture phenomenon *(3)*. The risk of rupture is higher in liver

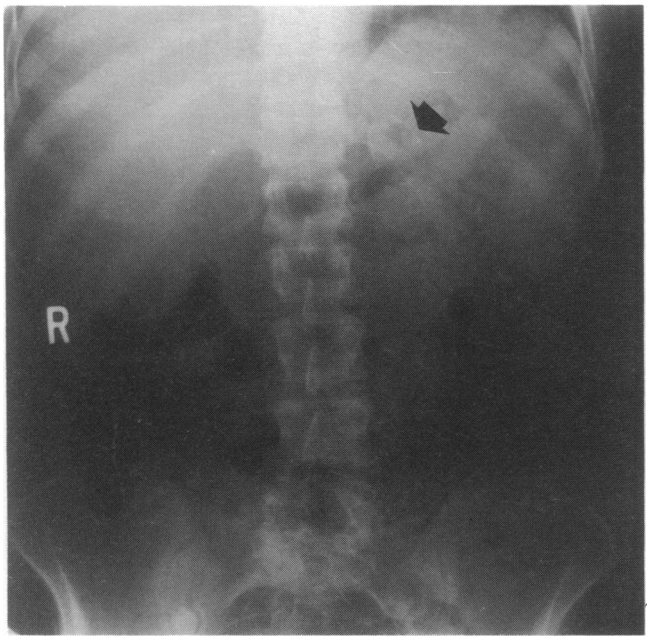


Figure 1. Plain radiograph showing the typical signet ring calcification of a splenic artery aneurysm.

transplant recipients *(47)*, pregnant women *(1–7, 50,51,68,72)* and large aneurysms *(2,69)*; however, most splenic aneurysms during pregnancy do not rupture *(1,9,68)*. The overall risk of rupture is about 20% *(3,52,68)*. Mortality after rupture reaches 25% *(1–7)*; during pregnancy, maternal mortality is 70% and fetal mortality is 75–90% *(1,3,27,28,68)*. Only 12 cases with fetal and maternal survival have been reported after rupture during pregnancy *(72)*.

In many cases the aneurysm can be evident on a plain radiograph, with vascular calcification forming a signet ring appearance *(1,3,5)* (Fig. 1). The diagnosis may be established by angiography (Fig. 2) or computed tomography (Fig. 3) undertaken for some other reason.

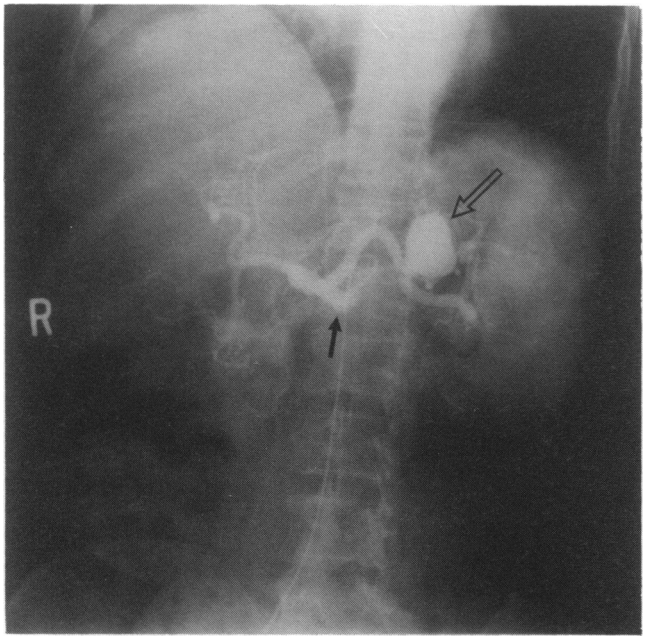


Figure 2. Selective coeliac angiogram showing a splenic artery aneurysm.

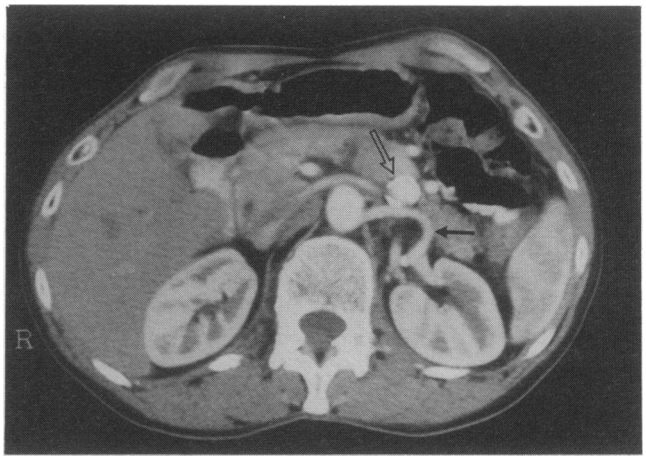


Figure 3. CT scan showing a splenic artery aneurysm.

Hepatic artery aneurysms (Figs 4,5)

They are usually solitary, fusiform or saccular and extrahepatic in about 80% of the cases *(3,7,12,41)*. Very few are symptomatic (acute epigastric discomfort due to expansion, similar to the pain of pancreatitis). Occasionally they may cause jaundice *(10,37)* or haemobilia *(11)*. Some 20% to 44% present with rupture, in which case mortality reaches 40% *(1–3,5,10–12,37)*. Rarely they are discovered by plain radiographs, because calcification is rare *(1)*. Most are discovered incidentally by angiography, CT scan (Fig. 4), ultrasound or MRI *(38,39)*.

Superior mesenteric artery aneurysms (Fig. 6)

They are usually situated at the proximal part of the artery, near the origins of the pancreatoduodenal and middle colic arteries, so the distal mesenteric circulation is compromised if thrombosis or dissection of the aneurysm occurs *(1–3,26,62)*. Rupture is unusual *(1,3,19)* and they are more likely to thrombose, therefore intraperitoneal haemorrhage is usually attributed to intestinal infarction *(1–3,5,13,19)*. The fact that almost 60% are mycotic *(3,13,19,48,64)* contributes to development of symptoms

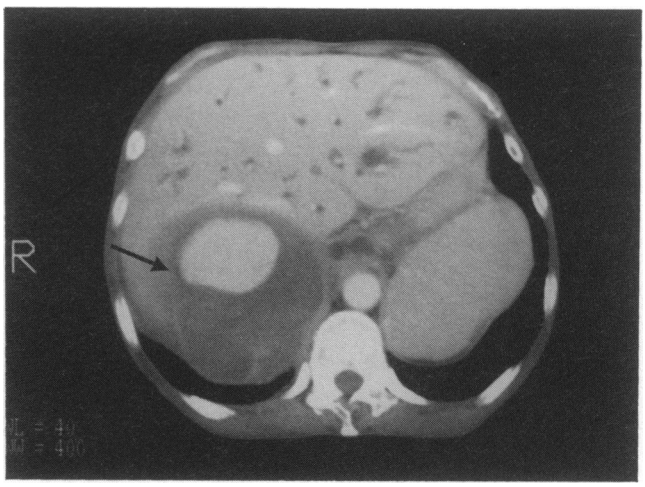
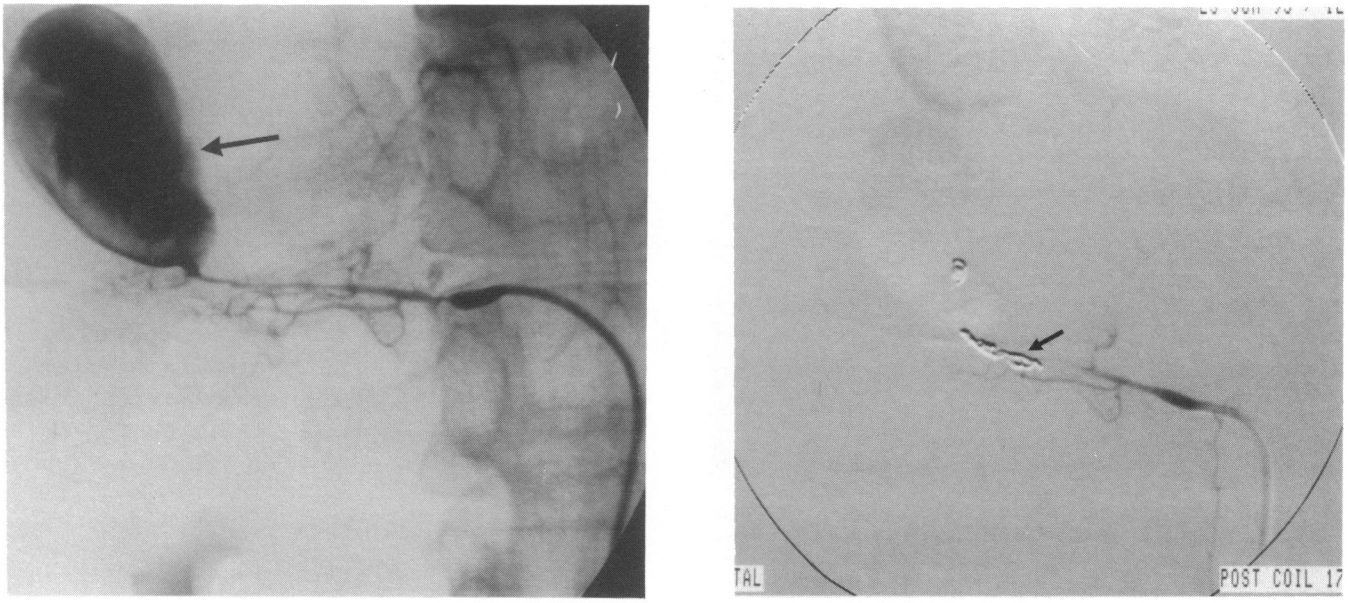


Figure 4. CT scan showing a huge intrahepatic aneurysm.



(*a*) (*b*)

Figure 5. (*a*) Selective hepatic angiography for embolisation of the artery from which the aneurysm originates. (*b*) Result after embolisation.

in the majority of these aneurysms. Abdominal discomfort varies from mild to severe pain and is commonly associated with intestinal angina. Mortality reaches 50% in cases of rupture or thrombosis *(1,26)*. In non-ruptured aneurysms the diagnosis can be established by ultrasound, CT scan or angiography *(1–6,27)* (Fig. 6).

Coeliac artery aneurysms

These are usually saccular, affecting the distal third of the vessel. Previously they were symptomatic because the majority were mycotic *(7,19)*, with an 80% rupture rate *(3,7)*, however, most recent series report a rupture rate of 13% with a >50% mortality rate, owing to exsanguinating haemorrhage *(1,6,7,26)*. The majority are diagnosed as incidental findings during other investigations or at surgical exploration.

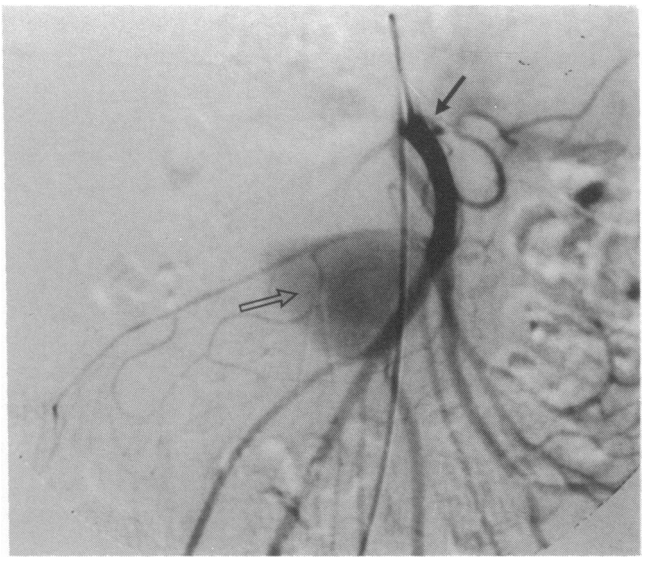


Figure 6. Angiography showing an aneurysm of the superior mesenteric artery.

Gastric and gastroepiploic artery aneurysms

Most are solitary and acquired, affecting men, aged over 59 years, three times more often than women *(1)*. Approximately 90% present with rupture *(1–3,26,48)* either into the peritoneum or the stomach *(7,20,26)*, where the mortality reaches 70% *(1,26)*. Rarely, they may be incidental findings on CT scan *(48,67)*.

Pancreaticoduodenal and gastroduodenal artery aneurysms (Fig. 7)

Most of these result from pancreatitis (60%) *(21,26,71)*, therefore, asymptomatic aneurysms of these arteries are unusual *(1,21–26,45,71)*. The majority of patients experience epigastric pain and discomfort, which may be related to underlying pancreatic disease *(21–25)*. Rupture may affect 50–75% *(21–26,48,71)* of these aneurysms with intraperitoneal bleeding being less common *(48,71)*. They usually rupture into the stomach *(22,45)*, the bile or pancreatic ducts *(24)* or a pseudocyst *(25,71)*. Mortality from rupture approaches 50% *(1–3,45,71)*.

Mesenteric artery branch aneurysms

Jejunal, ileal, colic and inferior mesenteric artery aneurysms more often affect elderly patients and may be multiple in 10% of cases *(26,28–31,43)*. Most of them are asymptomatic, but may present as either intraperitoneal or gastrointestinal bleeding *(28,30)*. Mortality during rupture is approximately 20% *(1–6,28)*. Angiography is the only means of diagnosis either before *(3,28)* or after rupture *(31)*.

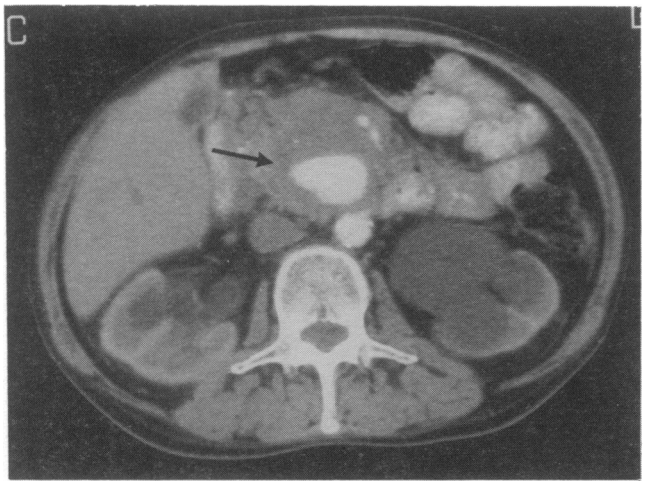


Figure 7. CT scan showing a false aneurysm of the pancreatoduodenal artery (after pancreatitis).

Renal artery aneurysms (Fig. 8)

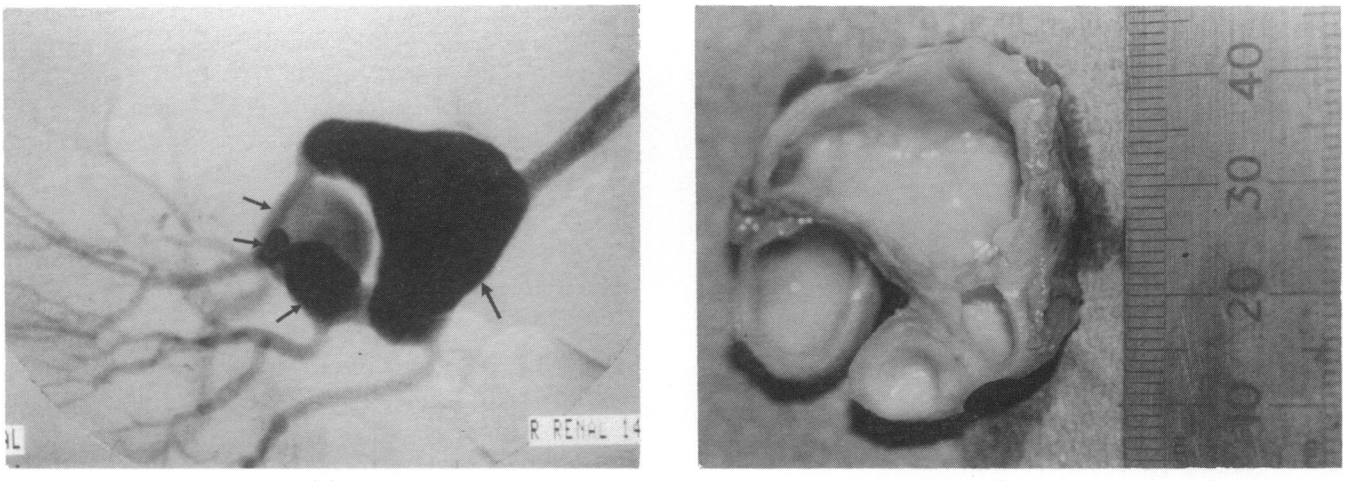
Improvement in imaging techniques in the past decade has led to increased recognition of true aneurysms of the renal arteries *(33–36,58)*. However, the natural history is not well known and considerable controversy exists about their clinical importance *(3,32–36,58,73)*. The right renal artery is affected more often than the left, which may be related to the greater incidence of fibrodysplasia on the right side *(32,60,65,66)*. Most are saccular, located in the primary or secondary bifurcations in 75% of the cases (Fig. 8*a*), while intraparenchymal aneurysms occur in less than 10% *(3,36,65,66)*. Most are asymptomatic *(32–35)*; however, the majority of reported aneurysms are related to hypertension *(3,58,74)*. About 30% develop symptoms *(56)*, including intractable hypertension, pain from expansion, haematuria, renal infarct and rupture. In a recent series *(74)*, 35% were detected during investigation for renal hypertension, whereas 26% were discovered incidentally by angiography undertaken for some other reason. Rupture represents the most serious complication and has been reported to approach 30% *(65)*; however, contemporary series show that the risk of rupture is probably less than 10% *(32,33,36,58,74)* and is related to hypertension *(75)*. In five series with a mean follow-up from 3–10 years, no acute rupture occurred during follow-up *(32,33,36,58,74)*. Mortality of rupture is also around 10%, but loss of the kidney is almost inevitable *(32)*. As with splenic aneurysms, rupture during pregnancy is a life-threatening condition, with maternal mortality around 50% and fetal mortality of 80% *(69,73,76,77)*. It has been suggested that asymptomatic aneurysms smaller than 1.5 cm with circumferential calcification are unlikely to rupture *(32,58,75)*, while those which are large and non-calcified, especially when accompanied by hypertension, are associated with a >20% risk of rupture *(58,65,75)*.

Treatment

Treatment options depend on the presence of symptoms, the relative risk of rupture, the mode of presentation and the anatomical location of each particular lesion. For most asymptomatic ones, simple observation usually suffices. However, for large aneurysms (>2 cm in diameter), perigastric aneurysms and aneurysms in women of child-bearing age, some form of intervention is necessary. Available treatment methods are embolisation, ligation of the aneurysm, resection procedures or endoaneurysmorrhaphy with some form of arterial reconstruction to establish the continuity of the involved vessel. For aneurysms presenting with rupture the main aim is to save the patient by controlling the bleeding and preventing related complications, such as distal organ ischaemia.

Splenic artery aneurysms

Large aneurysms and aneurysms recognised during pregnancy, even though asymptomatic, are a serious risk and should be treated, as the risk of mortality from



(*a*) (*b*)

Figure 8. (*a*) Renal angiogram in a patient with neurofibromatosis. There are four separate hillar aneurysms. (*b*) The specimen after surgical resection (with *in situ* arterial reconstruction).

rupture exceeds the perioperative mortality (0.5%) *(3)*. As most of them are solitary and in the distal third of the artery, splenectomy is the simplest and safest treatment option *(2,3,52)*. However, if splenic preservation is preferred, simple ligation or excision of the aneurysm may be performed *(78)*. False aneurysms related to pancreatitis are best treated by aneurysm incision and ligation of the vessels from within *(6,71)*. When intervention is necessary in high risk patients, percutaneous embolisation of the artery is a reasonable alternative *(2,3,52,79–81)*. Despite the fact that embolisation carries less morbidity than surgery and allows preservation of the spleen and its function, the risk of late rupture or abscess formation *(52,48,71)* suggests that it cannot be considered as the treatment of choice for young patients and those fit enough to undergo surgery.

Hepatic artery aneurysms

In proximal aneurysms, simple ligation without reconstruction seems to be adequate in most cases *(10)*. Liver ischaemia is more likely to occur in aneurysms of the distal part of the hepatic artery *(26)*. Trial occlusion of the liver circulation clarifies the need for arterial reconstruction with the use of a vein graft *(2,10,82)*. For aneurysms presenting with haemobilia or intrahepatic aneurysms, liver resection should be considered, but percutaneous embolisation (Fig. 5*a,b*) seems to be the most appropriate treatment *(2,79)*.

Superior mesenteric artery aneurysms

Simple ligation may be effective in some cases, but because of the risk of intestinal ischaemia and the propagation of infection, some form of reconstruction is usually employed *(1–6)*. This may be aneurysmectomy with end-to-end anastomosis or aortomesenteric bypass using autologous vein or artery as the graft material *(2,3)*.

Coeliac artery aneurysms

Surgical treatment is advocated for all coeliac artery aneurysms if the surgical risk is acceptable *(3,7)*. However, this could be a complex procedure, occasionally requiring a thoracoabdominal incision, with either graft interposition after aneurysmectomy or implantation of the coeliac artery to the aorta. There are reports showing a >90% success rate after surgical treatment *(7)*.

Perigastric aneurysms

As most of these (90%) present as emergencies without preceding symptoms, treatment is difficult. The catastrophic haemorrhage that follows rupture suggests that surgical treatment should be advocated even in asymptomatic aneurysms. No vascular reconstruction is necessary; simple ligation or partial gastrectomy for the intramural aneurysms is the treatment of choice. Embolisation is an alternative for high-risk patients *(79)*.

Surgical treatment is also mandatory for all false aneurysms of the gastroduodenal and pancreaticoduodenal arteries after pancreatitis. This is best accomplished by direct entry into the aneurysm and ligation of the feeding vessels from within *(71)*. Embolisation may also be tried *(79,80)*, but the risk of late rupture and abscess formation is significantly high *(3,44)*.

Jejunal, ileal, colic and inferior mesenteric artery aneurysms

As the risk of rupture is about 30%, surgical intervention is recommended. Arterial ligation is usually adequate but, for intramural aneurysms, excision of the involved segment of the intestine should be performed. Inferior mesenteric artery aneurysms can be treated by reimplantation of the artery to the aorta if the collateral circulation to the left colon is inadequate.

Renal artery aneurysms

Control of hypertension is associated with a lower incidence of rupture and is essential to all patients *(75)*. Surgical treatment should be used for all symptomatic patients. For asymptomatic cases, intervention is recommended for large aneurysms *(58)*, aneurysms that contain thrombus *(3)* or those which are associated with renal artery stenosis *(58)*. Finally, surgery should be undertaken for aneurysms in women of child-bearing age *(3,73,83)*. Surgery should remove (Fig. 8*b*) or exclude the aneurysm without compromising the kidney *(82)*. However, in the management of ruptured aneurysms, nephrectomy is the simplest and safest solution. *Ex vivo* repair is a safe technique and gives excellent results in most series *(58,74,84)*. For aneurysms of the proximal part of the artery, *in situ* reconstruction with vein grafts is the treatment of choice *(32)*. For intraparenchymal aneurysms, embolisation is probably the best option *(3,79,84)*. Recently, endovascular techniques with percutaneous insertion of graft stents have been employed *(85)*, but longer follow-up is required to assess the cost:benefit ratio. However, with refinement of the technique and increasing experience this promising technique may be used for all visceral aneurysms in the future.

Outcome

Good results have been reported with elective repair in all types of visceral aneurysms, with the exception of coeliac, superior mesenteric and hepatic artery aneurysms, which require more complex arterial procedures and are associated with higher mortality and morbidity. Nevertheless, surgical mortality is low, and if the correct indications for intervention are present, then surgical mortality is less than the 20–30% mortality associated with rupture of these visceral aneurysms. For poor-risk patients or for aneurysms in which arterial ligation would not compromise the more distal circulation, endovascular techniques represent a reasonable alternative.

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